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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles related to periodontology that appeared during the year of 1944 will be published in later numbers.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided by contributing the material for this issue.

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[*Reprinted from The British Dental Journal, 1944*]

The Underlying Principles of Dentistry*

. . . In view of the momentous decisions that must be taken by the profession in the near future, it would seem highly desirable that some precise statement should be made as to the nature and scope of dentistry. I venture, therefore, to submit a definition, and some deductions therefrom, to the members of the Association** for their consideration and criticism.

Definition.—"Dentistry is an art based upon a science. It is an integral part of the Health Service of the community, being concerned directly with the teeth and the surrounding oral tissues, and indirectly with the whole of the rest of the body."

Assuming that the statement above is correct, the activities of dentistry may be placed in three groups:

Group 1. The Tissues of the Teeth Proper.—In this group the dentist is dealing with tissues *sui generis*, specialized tissues unlike any others in the body. All loss of substance, whether as a result of operation or disease, must be restored by fillings, etc., and there is no repair by scar tissue. Dentistry in this group is a specialized profession, a profession *sui generis*.

Group 2. The Oral Tissues Surrounding the Teeth.—These are of the same nature as the tissues which go to make up the rest of the body, and are subject to the same natural laws. Their diseases come under the three main headings of the diseases with which medicine is concerned, viz. inflammations, degenerations, and neoplasms or new growth, and repair in all these diseases is by scar tissue. In Group 2, dentistry may be appropriately described as functioning as a branch of medicine.

Group 3. Dental Pediatrics (including Orthodontics) and Prosthetics.—The faculties and training specially needed here are those of an engineer, artist and craftsman. Nevertheless, this activity can only be exercised adequately by one who has also received the training necessary to discharge the functions referred to in Groups 1 and 2.

Finally, the whole of dentistry must be based upon the art and science of oral hygiene, since oral hygiene is the dental aspect of preventive medicine. I have never known of a really first-rate practitioner of conservative dentistry whose work was not built on a foundation of oral hygiene.

This conception of dentistry has been of the greatest assistance to me in endeavouring to assess the value of the various factors to be found in all dental problems. I trust that it may prove of equal service to others.

* A letter to the Editor of the *British Dental Journal* from A. E. Rowlett, Leicester, England

**[British Dental Association.]

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FORTY-FIVE

Report of A Case of Desquamative Gingivitis and Lichen Planus

By DANIEL E. ZISKIN, D.D.S. *and* HERBERT F. SILVERS, D.D.S. *New York City**

A FEMALE, white, age 38 years, presented complaining of a feeling of stiffness, burning, and tenderness of the cheeks, floor of the mouth, and limitation of tongue movement because of the soreness. Hot foods were also a source of irritation.

Medical History: Negative.

Dental History: The patient first noticed inflamed (desquamated) areas on the gingivae above both centrals and left lateral incisors, about three months prior to her first visit to our clinic. Lesions presenting clinical characteristics of lichen planus were observed on the cheeks, floor of mouth and palate at this examination. She had been using various mouth washes which were discontinued at that time, and she was put on a regime of vitamin B complex therapy consisting of 3 capsules daily for 45 days. Mild salt solution (½ teaspoonful in tumbler of water) was used during this period as a mouthwash. This therapy produced no symptomatic improvement. On a subsequent visit, it was discovered that many new areas of desquamation had appeared. The lichen planus lesions remained unchanged. The patient continued to complain of pain and sensitivity especially to hot foods.

Clinical Oral Examination: Our first examination in connection with this report was made about 6 months after the onset of symptoms. A biopsy was taken at this time from affected areas in cheek and on the gingivae, and follow up specimens were obtained at the end of 1 year and 2 years. All previous treatment was discontinued. Amalgam restorations were present in most of the posterior teeth and silicate fillings in the anteriors; the upper right and lower left bicuspids were non-vital; the upper right lateral incisor was replaced by a fixed bridge; several posterior teeth were missing and had not been replaced.

At this visit, we observed bright red areas on the upper labial and buccal gingivae over the right and left central and lateral incisors and cuspids; the upper left first bicuspid; upper right first molar; and on the lower labial and buccal gingivae around the left bicuspids and molars; and lower right bicuspids (Fig. 1). We also observed lichen planus-like lesions which covered the inner surfaces of the cheeks, muco-buccal folds, hard and soft palate, lateral borders of the tongue and floor of the mouth, and mucous membrane of the lips (Figs. 2 and 3).

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Fig. 1. Pretreatment photograph shows desquamated areas in upper anterior region.

Fig. 2. Pretreatment photograph shows lichen planus lesions in cheek—reticulated pattern.

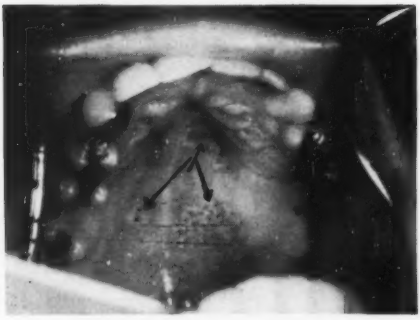

Fig. 3. Pretreatment photograph shows three separate areas of lichen planus on palate.

Fig. 4. Photograph taken 17 months after beginning of treatment shows that areas of desquamation have remained healed.

Fig. 5. Photograph taken 17 months after beginning of treatment shows that lichen planus of cheek has disappeared and not returned. (13 months vitamin A therapy).

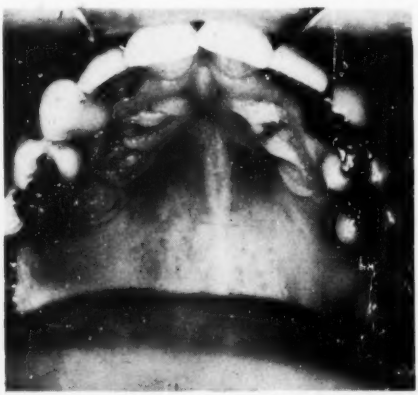

Fig. 6. Photograph taken 10 months after beginning of treatment shows lesions in palate have disappeared. (6 months vitamin A therapy).

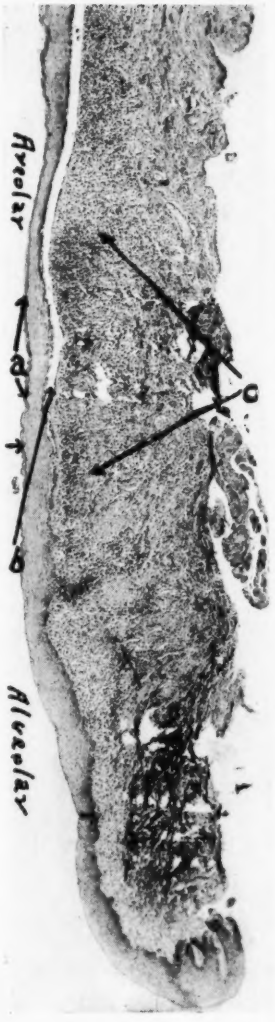

Fig. 7. Pretreatment biopsy of upper left #1 interdental papilla shows atrophic epithelium, intense inflammatory reactions and stripping of epithelium. The alveolar and areolar portions include: (A) keratin on areolar, (B) stripping, (C) intense inflammatory exudate. Mag. X 32.

Fig. 8. Biopsy of upper left #1 interdental papilla taken 13 months after that in Fig. 7. It shows reestablishment of peg pattern, healing of basal layer, and marked increase in thickness of epithelium and connective tissue. Inflammatory exudate is still present. There is a lessening of the tendency of the epithelium to strip in handling. Mag. X 31.

Fig. 7. Fig. 8.

Microscopic findings: (Upper left central incisor papilla). The whole papilla was atrophic (Fig. 7). The epithelial peg pattern had disappeared. In the alveolar gingiva, the keratin was somewhat reduced in thickness, but a layer of parakeratosis about 2 cells deep was seen under it. In the areolar gingiva, a well defined keratin layer was seen extending on to the mucous membranes (Fig. 9). (Keratin is not ordinarily found in this area). Under this keratin layer a few granulosa cells were seen. The epithelium was much reduced in thickness throughout, while in some areas it was exceedingly thin. Inter- and intra-cellular edema was seen throughout. A dense zone of inflammatory exudate composed chiefly of round cells, was seen in the papillary layer throughout the entire section. The edema in this region was quite marked and in some places, the epithelium could be seen stripping off from the corium. Inflammatory exudate

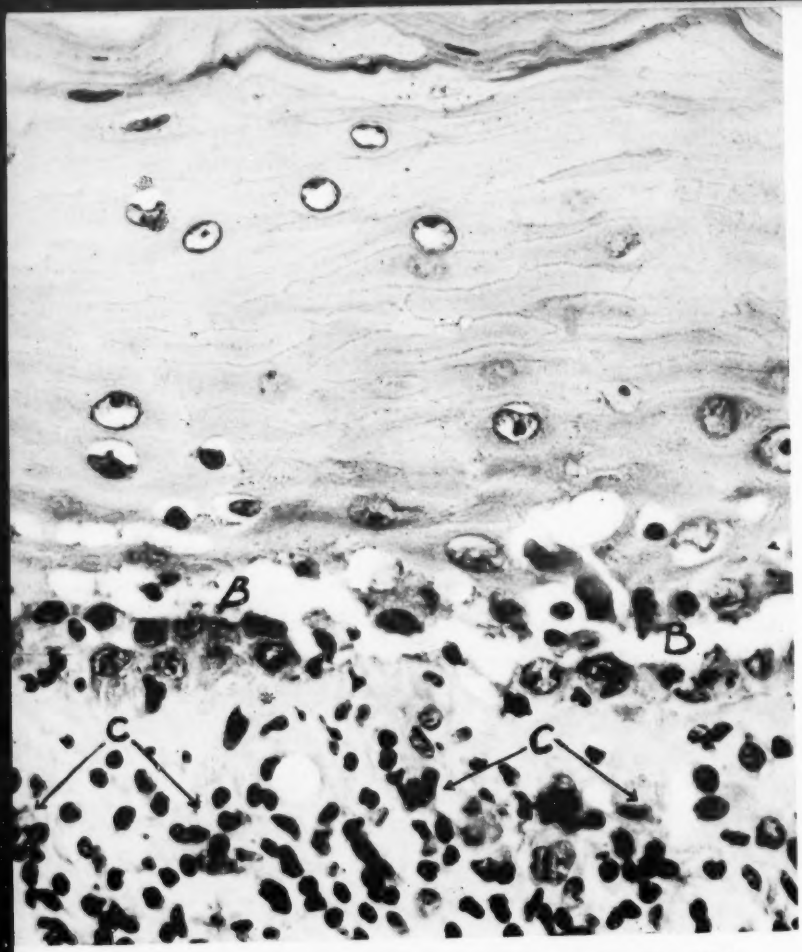

Fig. 9. High power of tissue in Fig. 7 (areolar portion), (A) keratin, (B) subepithelial edema and necrosis of basal cell layer, (C) inflammatory exudate. Mag. X 640.

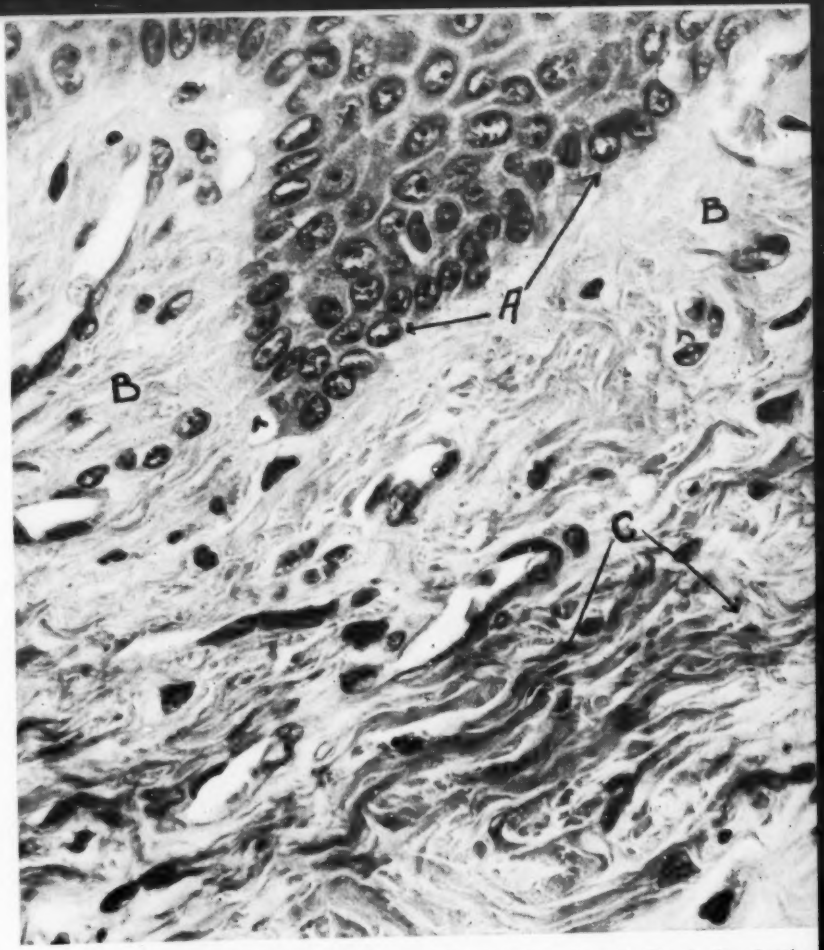

Fig. 10. High power of Fig. 8 (areolar gingiva). Compare with Fig. 9. The basal cell layer (A) appears normal. The collagen is dense and inflammatory exudate and edema have largely disappeared (B), (C). Mag. X 650.

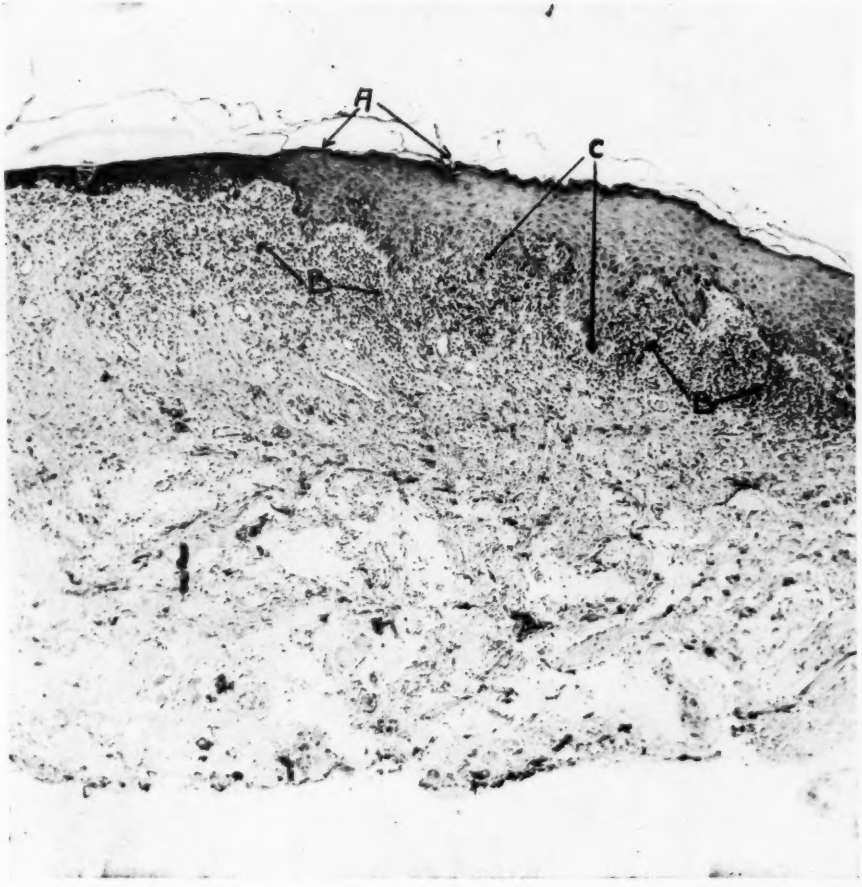

Fig. 11. Section from right cheek before vitamin A therapy was started. Taken after 4 months of estrogen therapy, it shows at (A) keratin not normally present, (B) inflammatory exudate, (C) edema and destruction of basal cell layer. Mag. X 65.

and edema extended deeply into the connective tissue.

The basal cell layer had also undergone atrophic degeneration. Widespread necrosis could be seen along this layer. Inflammatory cells had invaded the epithelium. In some areas, the epithelium appeared to be compressed.

In the corium, the vascularity was markedly increased. The collagen appeared spongy and granular. Fibroblasts and fibrocytes were seen, but these were almost obliterated by the denseness of the inflammatory exudate.

Cheek: This tissue appears to follow quite closely the description already given for the gingivae. Keratinization is seen throughout as a uniform dense layer. More of the peg pattern was seen here than was seen in gingival sections. The inflammatory zone was more confined to the papillary layer. Edema of the epithelium was not as marked, although present.

The basal cell layer did not present the same degree of destruction although some necrosis was present. The difference between the cheek tissue and the gingivae seemed to be mostly in degree rather than in kind of change (Fig. 11).

Treatment: Our treatment consisted of

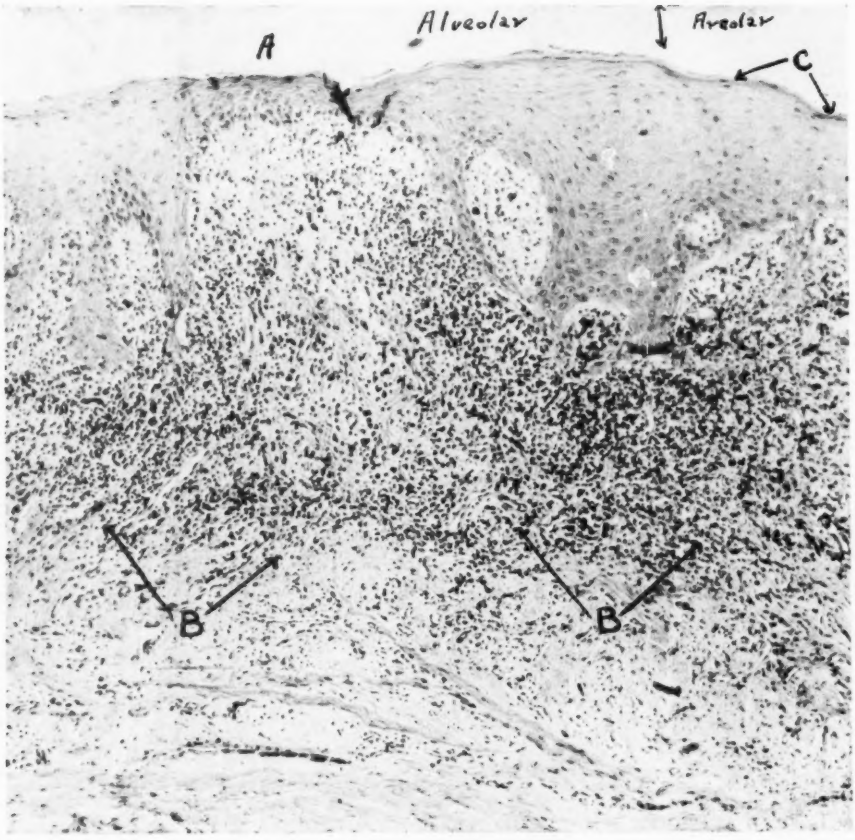

Fig. 12. Pretreatment biopsy of the lower left #3 interdental papilla shows section of alveolar and areolar tissue, (A) area of almost complete denudation of epithelium, (B) intense inflammatory exudate, (C) keratin in areolar portion. Mag. X 94.

the topical application of an estrogenic ointment* (estradiol benzoate). The method of applying the ointment was as follows: Shellac base plate trays (upper and lower) were made which overlapped the teeth and gingivae. These were filled with the estrogen ointment so that the salve was kept in contact with the gingivae, cheeks and floor of mouth for about 45 minutes. This method of application was repeated once each week for 25 consecutive weeks, and was augmented by home treatment when patient "buttered" the ointment on all affected parts at least once a day, usually before retiring.

The dosage can be only roughly estimated because there was no way of determining how much of the hormone was absorbed. A considerable quantity of the ointment was lost through drooling so that only a very small part of the applied hormone could have been absorbed. About 4,000 rat units were applied by us at the weekly treatments. Each tube of ointment prescribed for home use contained 25,000 rat units of estradiol benzoate and lasted for one week with the method of application described above.

The estimated dosage over a period of a year was about 1,500,000 rat units applied. The amount absorbed was not enough to disturb the menstrual cycle. However, while the systemic effect was probably negli-

* Progynon DH ointment (Schering).

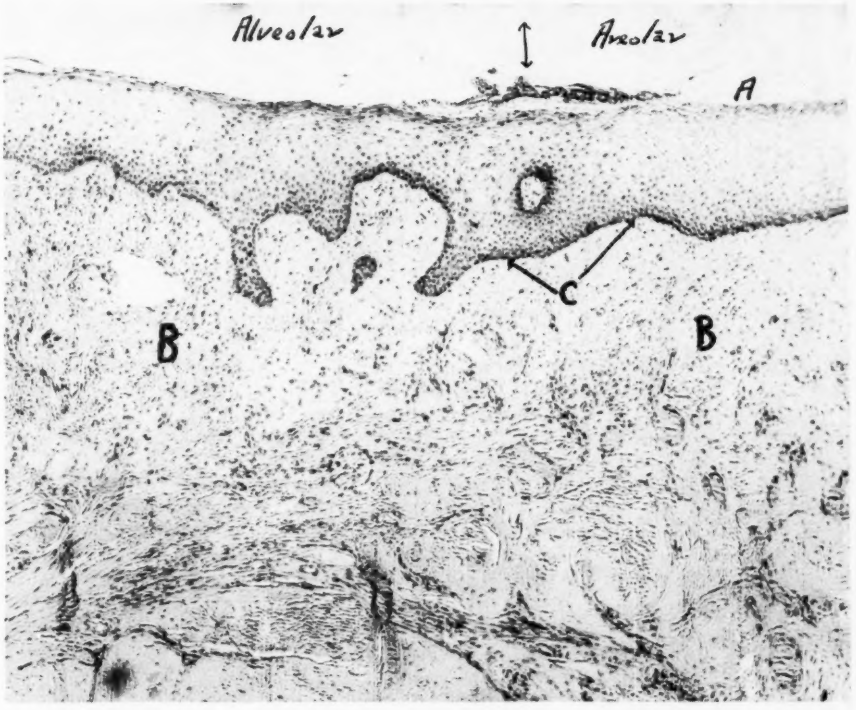

Fig. 13. Lower left #3 interdental papilla taken about 2 years after that in Fig. 12 shows alveolar and areolar junction, (A) absence of keratin, (B) firm connective tissue with almost complete disappearance of inflammatory exudate, (C) definite basal cell layer. Mag. X 80.

gible, the action, locally, was sufficient to produce the desired effect.

Three weeks after the above therapy was started, clinical signs of patches of epithelium, resembling tiny grafts, were noted on the desquamated areas. Six weeks after starting the treatment, there was obvious improvement. The epithelium formed a continuous layer over the formerly desquamated areas. Four months after the beginning of treatment, careful tooth-brushing was started. Use of a rubber interdental stimulator was also instituted at this time. About 500,000 rat units of estrogen ointment had been applied up to this time.

The lichen planus-like lesions had not changed appreciably, clinically, with this method of treatment except that the highly inflamed centers had become noticeably paler. The estrogenic hormone applications at home were continued, and in addition we prescribed one capsule of vitamin A in oil (25,000 I.U.) by mouth daily. After two weeks, since no improvement was seen, the dosage of vitamin A was increased to 50,000 I.U. daily. Four months later, 100,000 I.U. units of vitamin A distolate in sesame oil was injected intramuscularly once a week in addition to the above therapy.

The patient was now receiving 50,000 I.U. vitamin A by mouth daily and 100,000 I.U. intramuscularly once a week. She was also applying the estrogen ointment at home.

Five and a half months after starting the vitamin A therapy, the lichen planus lesions on the cheeks and palate had disappeared and intramuscular therapy was stopped. The patient had received by mouth and parenterally about 8,000,000 I.U. of vitamin A in all (Figs. 4, 5, and 6). The oral vitamin A was now reduced to one capsule of 25,000 I.U. three times a week. After three weeks of

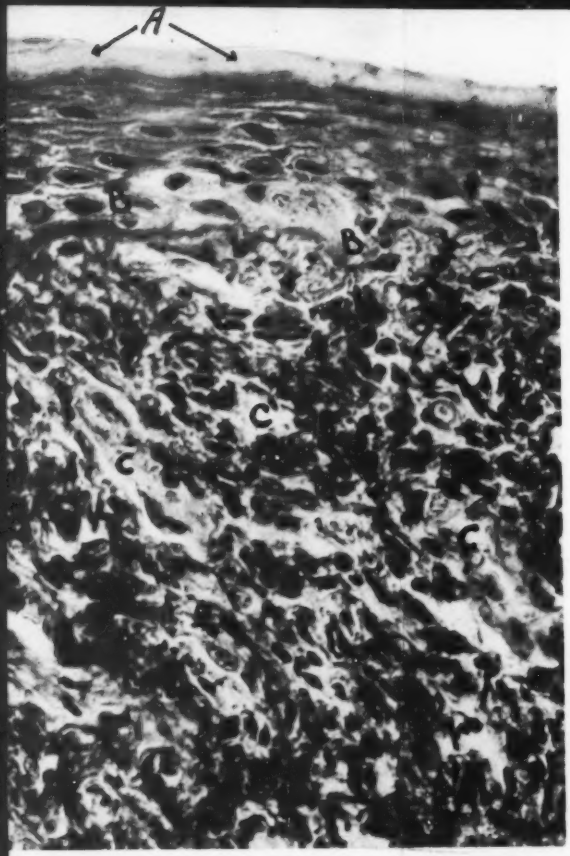

Fig. 14 High power of Fig. 12 shows (A) surface keratin in areolar portion, (B) destruction of basal cell layer, (C) intense inflammatory exudate largely obliterating the corium. Mag. X 490.

Fig. 15 High power of Fig. 13 shows (A) restoration normal unkeratinized areolar portion, (B) firm basal cell la and (C) normal appearing connective tissue. Mag. X 3

reduced vitamin A, both the subjective and objective symptoms of lichen planus reappeared in mild form.

The dosage of vitamin A was increased to 25,000 I.U. daily by mouth. After three weeks, the symptoms again disappeared. The medication was then maintained at this dosage. The patient was under observation for 14 months (to present time) after the second disappearance of the lesions. There has been no recurrence. The patient is still applying the estrogenic hormone ointment and taking one capsule of vitamin A (2500 I.U.) daily.

At this time, the desquamated lesions are healed. Clinically, a faint redness can still be seen in the areas which were formerly bright red over the upper left central and lateral incisors, even though covered with epithelium. There are no subjective symptoms.

Microscopic follow up: *One year* after the pretreatment biopsy of the upper left central incisor area, a second biopsy was taken (Fig. 8). This showed the epithelium to have regenerated considerably. The whole layer was deeper and the epithelial peg pattern had been reëstablished. The basal cell layer was firm and was attached securely to the corium having lost its tendency to strip off (Fig. 10). The connective tissue layer was also markedly increased in width. There was still a large amount of inflammatory exudate present in the deeper layers of the corium, the papillary layer being almost free. There was an increased vascularity and many fibroblasts were in evidence. Heavy collagenous bundles could also be seen in the corium and the edema had been greatly reduced. The abnormal keratin layer had almost entirely disappeared.

A follow up biopsy after *two years* of treatment was taken of the lower left cuspid papilla (pre- and post-treatment biopsies taken from same area) (Figs. 12, 13, 14, and 15).

It will be noted that there has been further reduction in the inflammatory exu-

date while the appearance of the epithelium remained normal. The collagen has also increased in density. The abnormal layer of keratin on the areolar portion has disappeared. The striking features of this section were (1) return of the mucous membrane to its normal unkeratinized state (2) the marked reduction of inflammatory exudate (3) the denseness of the connective tissue. It will be seen, however, that there was still some inflammatory exudate present.

DISCUSSION

This case presented two types of lesions from the clinical standpoint which are often seen alone. The fact that they appeared together indicated the possibility of a common etiology. When found singly, they respond to different forms of treatment just as they did in this case. In an attempt to evaluate the beneficial effects of each therapeutic agent, we found that using the estrogen alone did not materially benefit the lichen planus-like lesions. We did not try the vitamin A alone in this case because of our negative results in other cases of desquamative lesions. However, when the vitamin A was reduced to 75,000 units per week, the lichen planus-like lesions on the cheek mucous membranes and palate promptly returned. The subjective symptoms also returned.

In observing this case over a period of more than two years we are of the opinion that the vitamin A therapy aided the symptoms of the lichen planus-like lesions, while the estrogens were of benefit to the desquamated lesions. These observations point to a metabolic disturbance in the tissues. However, since no systemic involvement was apparent, the disturbance must be considered local in nature. The etiology is obscure. The interrelations involved in this case are more fully discussed in other reports. (1), (2).

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In Memoriam

LYNN A. FONNER

DECEMBER 7, 1944

Gingival Inclusions*

B. ORBAN, *Chicago, Illinois*

IN ROUTINE examination of biopsy specimens from the gingiva a variety of inclusions could be found. An inclusion is any foreign or heterogenous substance contained in a tissue, not introduced as a result of trauma. *(Steadman's Medical Dictionary)* Some of these inclusions can easily be recognized as tooth fragments consisting of cementum and dentin (Fig. 1). Such tooth particles embedded in the soft tissues might have different origins such as remnants of deciduous teeth or fractured torn pieces of the root surface of permanent teeth. In biopsy specimens, the origin of these bodies cannot be ascertained because, whatever the origin might have been, the microscopic appearance of dentin and cementum is the same whether it is of a deciduous or permanent tooth. However, in specimens of teeth sectioned together with their supporting tissues, the origin of these inclusions usually can be determined.

The numerous inclusions seen in Figure 2 are certainly remnants of the earlier extracted teeth in this area. They consist of ce-

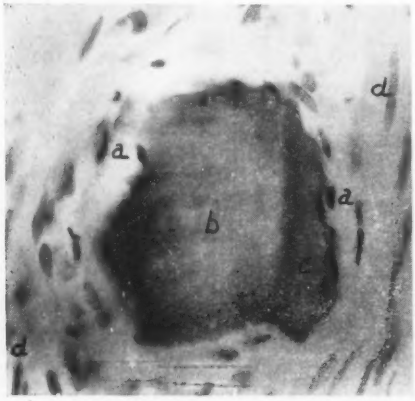

Fig. 1. Dentin-cementum remnant is a gingival biopsy specimen. Osteoclasts (a) (mononuclear) resorb the surface of dentin (b) as well as cementum (c). Fibrous capsule (d) surrounds the inclusion.

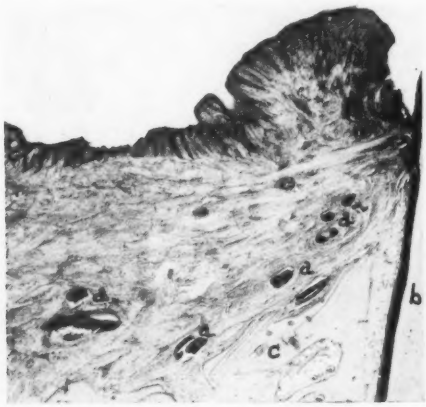

Fig. 2. Numerous dentin-cementum splinters (a) in the area of previously extracted lower central and lateral incisors. One central incisor seen in picture (b). All splinters are above the bone (c). Extraction wound completely healed.

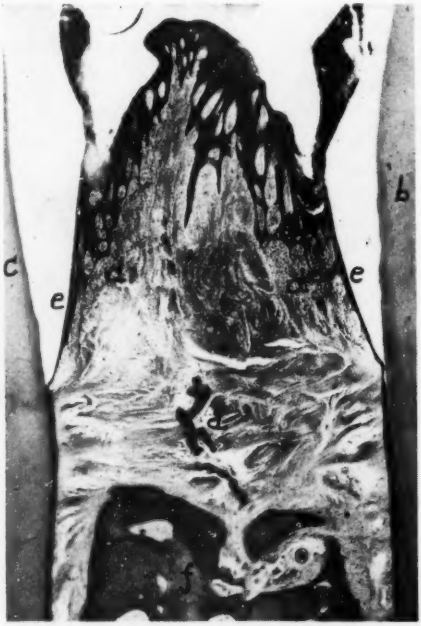

Fig. 3. Cementum remnants of a deciduous tooth (a) in the interdental papilla, above the alveolar crest, between upper lateral incisor (b) and cuspid (c). (Individual aged 19 years.) Inflammation in the gingiva of slight degree (d). Epithelial attachment on the enamel (e). Alveolar crest (f).

* Foundation for Dental Research of the Chicago College of Dental Surgery, Loyola University.

Fig. 4. Cementum fragments (a) and a cementicle (b) close to the gingival pocket (c) in the bifurcation of a lower molar. Resorption and repair on the root surface (d). The cementum fragments are surrounded by inflammatory granulation tissue of chronic character.

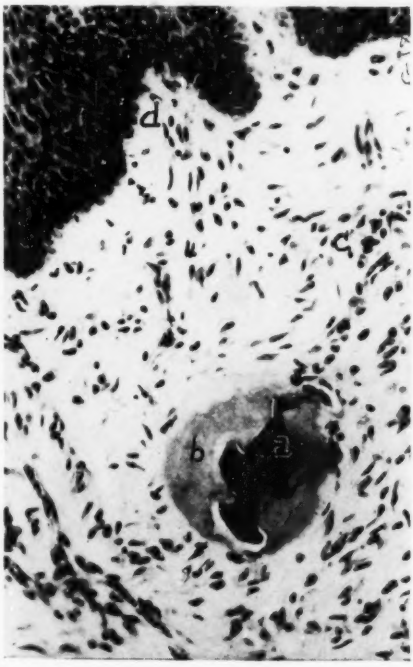

Fig. 5 (Left) Gingival biopsy specimen. Calcified tooth remnant (a) with resorbed surface surrounded by newly formed hard substance (b). Around this inclusion chronic granulation tissue (c) Epithelium of gingiva (d).

mentum and here and there some dentin. The extraction wound is completely healed and all splinters are found above the bone, some close to the tooth surface, some farther away.

In Figure 3, the inclusions seen in the interdental space, above the alveolar crest, are most probably remnants of a deciduous

Fig. 6. Cementicle (a) in a gingival biopsy specimen. No inflammation in this region. Well hornified epithelium (b). The space in the center of cementicle is an artifact.

tooth. These cementum pieces are between the upper lateral incisor and cuspid of a 19 year old individual. No teeth are missing and there are no signs of cementum fractures on the neighboring teeth.

The calcified bodies in Figure 4 have been

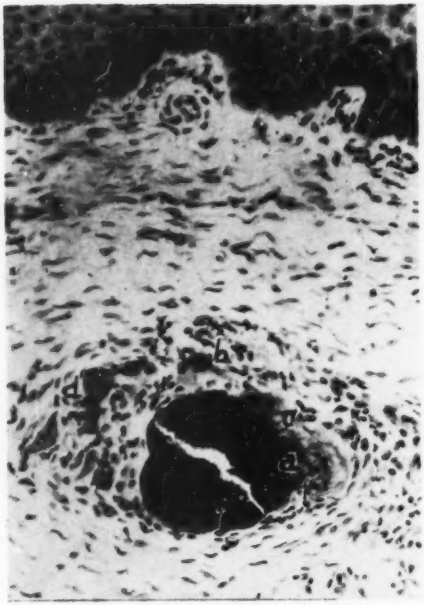

Fig. 7. Cementicle (a) (with artificial crack in the center) in a gingival specimen. Osteoclast (b); new formation of hard substance (c). Bone spicules (d). Chronic granulation tissue surrounds the inclusions.

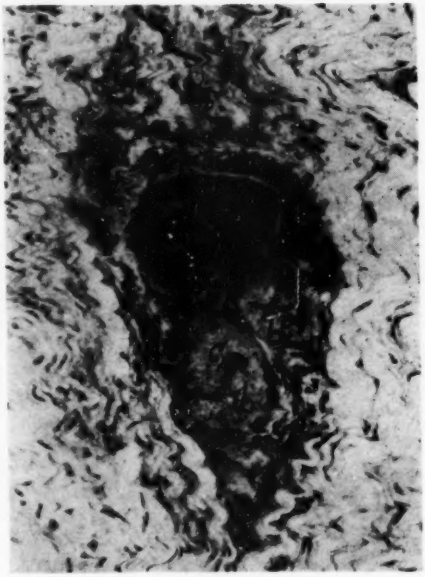

Fig. 8. Calcified cementicle-like inclusion (a) in a gingival biopsy specimen. Hyalin degenerated tissue (b) with calcified center (c) indicate further development of similar bodies. Calcium granules in the hyalin tissue. Fibrous capsule surrounding inclusions.

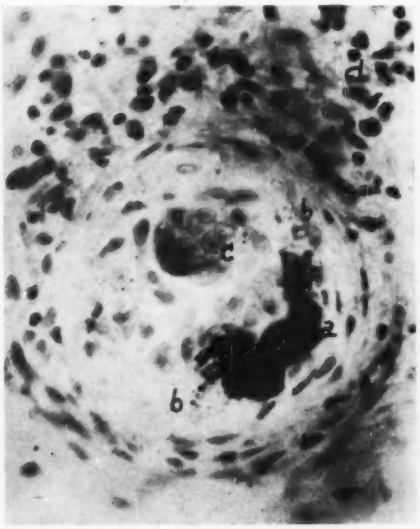

Fig. 9. Calcification of a blood vessel (a) in a gingival biopsy specimen. Hyalin tissue, with calcium granules (b). Giant cell (c). Chronic granulation tissue (d).

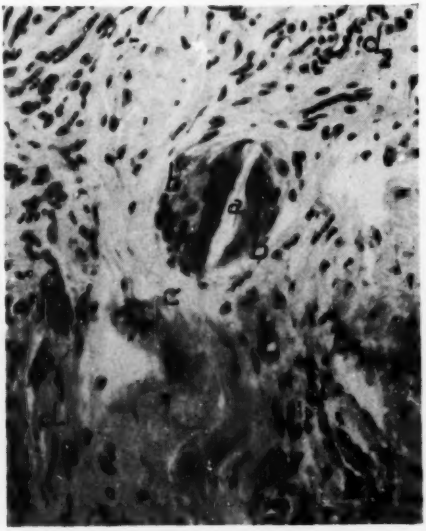

Fig. 10. Crystaline inclusion (a) surrounded by foreign body giant cell (b) in a gingival biopsy specimen. Fibrous capsule (c). Chronic granulation tissue (d).

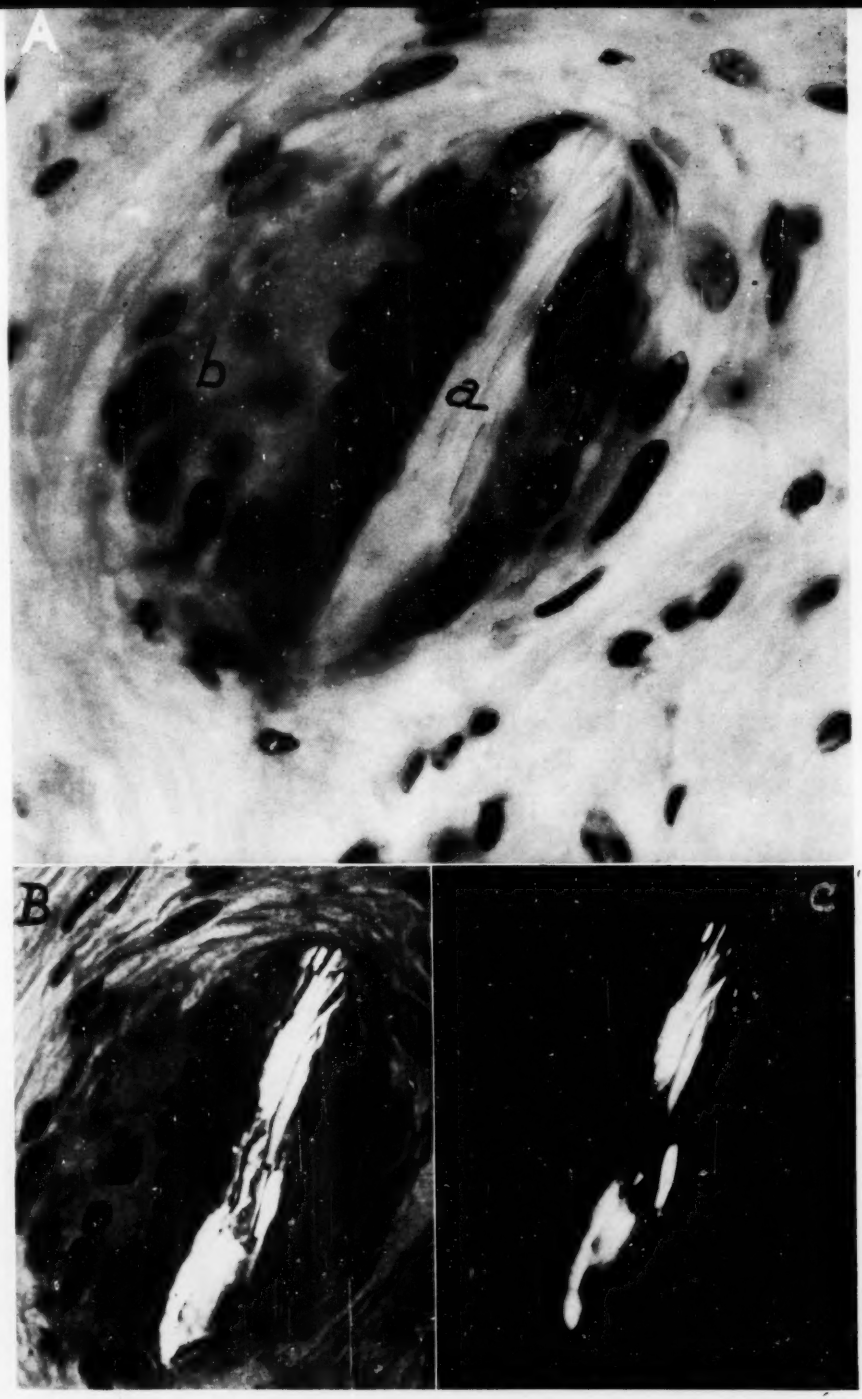

Fig. 11 A. High magnification of crystals (a) and giant cell (b) of the previous illustration. 11 B. By viewing the same area with polarized light (polaroid filters), and turning the occular 45 degrees, the tissue turns dark, whereas the crystals become highly refractile. 11 C. By turning the occular 90 degrees, the tissue completely disappears, but the crystals shine brightly.

found in the bifurcation of a lower molar. The surface of the cementum shows signs of former injury—probably tears. The cementum surface was later resorbed and then repaired by new cementum formation. The calcified bodies in the tissue are most probably remnants of cementum torn away in an injury. These show signs of resorption as well as new bone deposition. One of the calcified bodies is a dislocated cementicle frequently found in the periodontal membrane.

Such cementum and dentin remnants are frequently found, not only in the deeper tissue layers, but in the gingiva itself, and are therefore found also in biopsy specimen. They are frequently surrounded by a fibrous capsule. Sometimes resorption and repair processes alternate and we find the old tooth remnants enclosed within newly deposited hard substance, (Fig. 5) forming a round body. Not infrequently we find calcified bodies, resembling cementicles, in biopsy specimen of gingiva (Fig. 6). These inclusions show a cloudy, granulated appearance without any definite structure. There might be one or more of these bodies in a group. Frequently an artificial tear is seen crossing the center of the calcified body. Sometimes deposition of new hard substance can be observed on the surface, and in other inclusions giant cells appear to resorb the calcified body (Fig. 7). A few fine bone spicules lie close to this cementicle. From the observation of one specimen (Fig. 8) it seems possible that denticle-shaped calcified bodies might develop in the gingiva itself. A hyalin degenerated tissue with granules of calcium seems to form several round cementicle-shaped bodies. One body is already calcified, the other two have a calcified center with a large granulated outer layer and are most probably on their way to being calcified.

In one instance a thrombosed calcified blood vessel could be observed in the gingiva (Fig. 9). A giant cell appears also in this specimen.

In several specimens peculiar crystaline bodies could be observed, mostly surrounded by foreign body giant cells (Fig. 10, 11). These crystaline bodies appear, in the hematoxylin-eosin specimen, to be transparent,

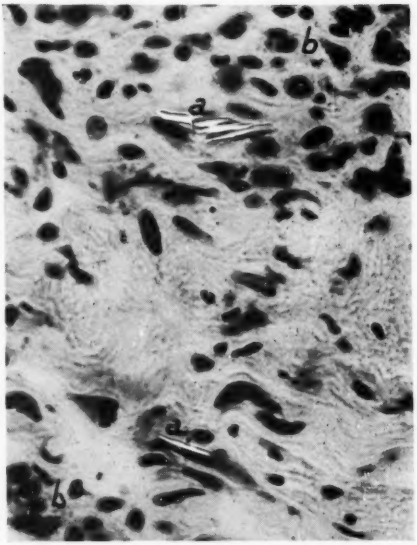

Fig. 12 Gingival biopsy specimen. Crystals (a) (most probably cholesterol) in chronic granulation tissue (b), made visible by polaroid filters.

light yellowish, and are hardly recognizable. In the polarized light they are birefringent, as seen in Figure 11B and C. Sometimes crystals, single or in groups, are found free in the tissues without a foreign body giant cell (Fig. 12). They are surrounded by chronic granulation tissue. Their origin is not as yet explained. It is known that crystals appear sometimes in inflammatory tissue and they are known as Charcot-Leyden crystals which develop from nuclein decomposition or from oxyphile granules of the eosinophile leukocytes.[1] It is possible that these crystals are of this nature. The possibility also might exist that they represent uric acid crystals as found in gout. However, it is most probable that they are cholesterol crystals which are occasionally found in areas of tissue decomposition.

It has been suggested that some microscopically small foreign bodies might enter the gingiva from a dentifrice, toothbrush, or in some other way. It is possible, but at the present time no material is available to prove it.

[1]Wells, H. G., *Chemical Pathology* (Philadelphia, W. B. Saunders Co., 1920) pp 311-312.

SUMMARY

1. Inclusions of different structures are frequently found in the gingiva.
2. These inclusions might be cementum fragments of shed, or extracted, deciduous teeth, or extracted permanent teeth.
3. Cementum fragments due to traumatic injuries may also be found in the soft tissues without any preceding extractions.
4. Cementicles from the periodontal membrane, dislocated during tooth eruption, might appear in the gingiva. Resorption and repair can take place on these bodies.
5. Cementicle-shaped calcified bodies might also develop in the gingiva as a result of hyalin degeneration of connective tissue and subsequent calcification.
6. Crystaline foreign bodies are also found in the gingiva. Their origin and nature is not clear, as yet, but they are most probably cholesterol crystals.

(180 N. Michigan Avenue)

Nutrition and Dental Health

By DOROTHEA F. RADUSCH, D.D.S., B.A., M.S.
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THERE IS much presumptive evidence and some specific evidence indicating that nutrition plays an important part in dental health and disease. Since research along these lines of special interest to dentists is generally published in widely scattered journals, it seemed desirable to review this pertinent information in the Journal of Periodontology to keep busy dentists up to date, as well as to give emphasis to the data by aggregation.

To give a proper basis for discussion of the special relationships between nutrition or diet and dental health or disease specifically, basic nutrition facts must be reviewed.

Probably no one factor affects our health so much today, as the food we eat. The Report of the United States Conference on Food and Agriculture, recently held at Hot Springs, Virginia, states that investigations show that the greater part of the diets of all peoples is nutritionally unsatisfactory; that this leads to impaired physical development, ill health, and untimely death; and that, through diet a new level of health can be obtained enabling mankind to develop inherited capacities to the fullest extent.

These conclusions apply to our own United States as well as to the rest of the countries which were represented at the conference. Every nutritional survey in the past decade has revealed that the consumption of diets below the recommended standards for satisfactory nutrition is wide spread in the United States. An appreciable percentage of the diets failing to meet the standards set by the National Research Council were more than 50 per cent deficient in amounts of the several essential nutrients; but most of the diets were less than 50 per cent deficient. We know too, that those regions of the United States known to be the worst fed contributed the highest ratio of rejections of the more than one-third of the military recruits which were found physically unfit for service.

Unquestionably there is wide-spread prevalence of moderately deficient

diets. It is this state of "moderately deficient" which is so insidious. Diagnostic methods for the detection of conditions arising from such moderately deficient states are not well established in the medical professions, nor is there sufficient appreciation of the importance of moderately deficient states which do not give rise to classic symptoms of deficiency diseases.

In the new conception of the meaning of nutritional well-being, an individual need not be disabled or nearly so by rickets, scurvy, pellagra, xerophthalmia, beri-beri or other recognized deficiency diseases to be considered poorly nourished. Malnutrition is not synonymous with deficiency disease. An obese individual is as truly malnourished as an undernourished one.

New objective methods for measuring nutritional status show that mild deficiencies exist in many people. Such measurements have been made along with dietary surveys of the same subjects. In one Pennsylvania study,* the 2400 subjects were studied twice, at two seasons of the year, and no persons were included who were adjudged by a medical examination to have a pathologic disorder which would interfere with proper nutrition. They were average people from homes, schools, factories, mines, stores, and professional offices, representing a large number of socio-economic and racial groups.

Only 16.8 per cent of all subjects were found in the optimum class with respect to the standards for his own age and sex in skeletal mineralization, 43.2 in hemoglobin, 59.4 in weight status, 7.3 in appearance of nutritional status by physical examination. In no income or educational group were even a majority of the subjects ideally nourished in the various respects covered by the tests.

A recent bulletin of the National Research Council (No. 109, Nov. 1943), "Inadequate Diets and Nutritional Deficiencies in the United States—Their Prevalence and Significance" brings together all available published and unpublished material of the result of dietary surveys and nutritional appraisal. Herein is recorded overwhelming evidence that deficiency states are present on a large scale in all income and educational levels; most of them not of the severe acute type, but rather, the less intense mild or moderate forms, which because of their slow gradual development are commonly unsuspected or undetected.

The earliest symptoms of malnutrition are often vague and most certainly are not diagnostic of any particular deficiency disease or of malnutrition itself. They include such conditions as poor appetite, easy or excessive fatigue, constipation, insomnia, irritability, neurasthenia, eye strain, sore tongue, paresthesias, and probably dental caries and periodontal disturbances. All these should be looked for and listed in any nutrition or diet survey, even though the investigator must be aware that these symptoms are *not specific* for malnutrition.

Then what are the signs or symptoms of malnutrition? Well established only are those for gross deficiencies, while the common dependence on height and weight measures is the least reliable of any measure of good nutrition. Specialized technics for detecting deficiency states are being worked out. A few are already well established, such as those for iron (hemoglobin), protein, and ascorbic acid. Data for others are still in the investigative stage. Experienced

* *Family Nutrition* (Philadelphia Child Health Society, Apr. 1942) Chap. II.

scientists can use certain specialized methods quite satisfactorily, which are as yet too involved for general use.

Too little use is made of actual diet records kept by patients. Members of the medical and dental professions assume that because a person has an adequate or good income, that he eats the proper food. Sometimes diet is discussed in general terms with the patient, but nutritionists know that little reliance can be placed on this type of dietary survey. Too many people do not realize that the citrus fruit juice "they like" actually has been eaten by them only once or twice a week instead of daily. Nor do they realize how infrequently they eat other foods needed to supply essential nutrients. Most patients even state that the diet record they have kept is not the usual intake, "oh, I usually eat better than that!" There is the excuse of being busy, having company, not feeling well, and so on. Actually, however, it is safe to assume that the written record is one of better than usual consumption, because the process of having to write down the food intake of itself makes the patient food conscious, and so he subconsciously improves the diet.

While inadequate consumption of foods supplying the essential nutrients is the most common cause of nutritional deficiency, it is recognized that there are other conditioning factors which may raise the requirements for the individual. These may be physical exertion, light, trauma, disease, pregnancy, and of course sex and age. Thus, without careful attention to all phases, deficiency states may occur even on a seemingly adequate diet. Consequently, higher prevalence of deficiency states could be expected even than those rates indicated by surveys of food consumption at a particular time.

The Bulletin, previously referred to, summarizes this as follows:

> But evidence that deficient diet or conditioning factors are in operation at some particular point of time gives only a slight and incomplete conception of the full scope of their influence and effects. The frequency and duration of their operation is extremely important. Thus time, which for persons is age, is a significant factor. Few people eat an adequate diet every day during their lifetime. Rather, there are numerous or protracted periods when the diet is faulty. Similarly, few people escape the operation of conditioning factors at frequent or lengthy periods in their life. The repeated or protracted operation of faulty diet or various conditioning factors slowly leads to cumulative effects in deficiency states.
>
> All the data from numerous surveys with new methods among persons of all ages in many regions are entirely in accord in showing that deficiency states are rife throughout the nation. Relatively few are the traditional severe, acute stages. Most are milder in intensity and gradual in their course. Predominantly they are subacute or chronic states: some marked, but very many mild or moderate.

In the evaluation of nutrition surveys (*Nutrition Reviews*, Jan. 1943, p. 76) it is stated:

> It seems reasonable to maintain that, although dietary studies do not directly measure the incidence of malnutrition, they furnish the best means now available to uncover likely areas of malnutrition before severe degrees of deficiency develop. They are therefore a most valuable public health tool. An inadequate diet stands in need of correction regardless of what evidence of deficiency disease the physician or biochemist is or is not able to uncover. Our ability to recognize the earliest stages of the deficiency disease is still markedly underdeveloped. In any event, it is hardly sound medical practice to wait until a person has developed a disease before attempting to correct the causative fault in the environment.

Greater attention should be paid by all to the clear convincing evidence of the relationship between faulty diet and ill-health and likewise, to the evidence of general improvement in health and physique which can be produced by the improvement of diets.

(Continued in the April issue.)

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Editorials

Periodontal Nomenclature

THERE are several reasons for bringing up this subject again, the chief one being that there are still too many clinicians, authors, and teachers who use the term "pyorrhea" to designate various forms of periodontal disease. "One cannot repeat too often what is not learned sufficiently". Disregarding the recommendation of the Committee on Nomenclature of the American Dental Association[1] that it (pyorrhea alveolaris) be discarded, many still use it.

The American Academy of Periodontology has had several well-qualified Committees on Nomenclature who have given the subject of periodontal nomenclature serious and thorough study. The first committee functioned at the time the Academy was organized, in 1914, and contributed the terms periodontology, periodontia, periodontist, periodontoclasia, and later periodontium, which have been generally adopted. Other Committees reported from time to time stimulating discussion but little action until at the 1937 Annual Meeting a most extensive Report was read and adopted[2]. A later Committee with more specific recommendations presented its Report at the 1940 Annual Meeting, where it was thoroughly discussed and adopted[3]. More recently, in 1943, the Nomenclature Committee of the American Dental Association in its Annual Report discussed *periodontal* nomenclature. This Committee defined among other periodontal terms the following of pure Greek origin: periodontium, periodontia, periodontist, periodontology, periodontitis, periodontosis, periodontoclasia. In the Report the Committee stated,

> Certain objectives in dental nomenclature seem to be desirable. If dental nomenclature is to be in keeping with the high classical and scientific esteem to which dentistry aspires, it should be of pure classical origins rather than made up of a collection of hybrid terms. Dental terms should not only be soundly developed etymologically, but should also be accurately defined. All scientific terms should preferably be fully informative and descriptive . . . . An exchange of ideas and thoughts between individuals is possible only by the employment of a language of marked specificity.

The Committee solicited the profession's support in order that unity of dental language be attained in this country. Since then most of the dental schools have seen the necessity of coöperating toward this objective and are doing so. It would seem therefore, that the least the individual clinician, researcher, and author can do is to accept and use the terms recommended by this Committee so that all will speak a similar language in periodontology.

One of the recommendations made by the 1940 Committee on Nomenclature and adopted by the Academy was to the effect that all papers published in the Journal of Periodontology be editorially revised "to comply with the official nomenclature". This recommendation has been only partially carried out because of a belief that a gradual change of policy was more desirable than an abrupt one. The time has now arrived for more positive action. In the future

[1] *J.A.D.A.* 30:1112. July, 1943; *J. Periodont.* Supplement to July issue, 1943. pp. 14-16.
[2] *J. Periodont.* 8(2):88-95. July, 1937.
[3] *J. Periodont.* 12(1):16-18. Jan. 1941.

all authors of papers published in this Journal will be asked to comply with this policy of the Academy. Many have done this in the past, especially those who have been eager to aid in clearing up the confusion of terminology in periodontal literature.

To avoid any misunderstanding of the official nomenclature, a series of short articles will be published in this Journal, beginning in the April, 1945 issue. These will be prepared by Dr. Harry J. Lyons, Chairman of the Committee on Nomenclature of the Academy. The sincerity of this dental teacher is well-known and his writings notable for their clear and explicit expression.

A common saying of the philosopher Myson was that, "Men ought not to seek for things in words, but for words in things". This idea is in agreement with several periodontists who have consistently contended that when once we fully understand the diseases that attack the periodontium it will not be difficult to name them. We can then find the correct name in the disease.

Meanwhile, in the interest of dentistry, the term "pyorrhea" should be discarded. The word, which means a flow of pus but does not designate its source, is used as one uses slang, from habit. It is employed mostly as a general term for periodontal diseases and often where no suppuration is present. Over the years dentists have used, then discarded inaccurate terms, for example, "dead teeth", "plates", "cleaning teeth", "silver fillings", "elongated teeth". Today, one rarely hears the term "straightening teeth". The orthodontists have corrected that. The oral surgeons have thrown out the expression "pulling teeth". The next step should be taken by the periodontists.

Dental patients can aid in the establishment and use of the official periodontal nomenclature. They quite commonly use medical terms such as tuberculosis, peritonitis, nephritis, poliomyelitis, pharyngitis, appendicitis, encephalitis, and endocarditis. The war has introduced many new terms which are readily used by people of average intelligence. Therefore, the old argument that the new periodontal nomenclature is too difficult in spelling and pronunciation to be generally acceptable, is no longer tenable.

Coöperation is what is needed at this time. "Coöperate. Advance." —G.R.S.

A New Journal Feature

THE reader's attention is called to the first of a series of articles on "Nutrition in Relation to Dental Health" by Dr. Dorothea Radusch, in this Journal. Dentists are finding an increasing need for knowledge of diet and nutrition to meet the demands for information from their patients, and also to satisfy their personal interest. World wars seem to be necessary to stimulate the serious interest of laymen in the subject, but researchers, nutritionists, and faddists consistently continue their investigations. Vitamins were new and but few had been discovered by the end of World War I. Today books are written on this phase of nutrition, alone. Because of the importance of the subject of nutrition and its relation to dentistry, a section of the Journal will be regularly devoted to it. Dr. Radusch is well-qualified by experience and research to sift the chaff from the wheat of available information in this field. This will be a real service to our readers.

The Physiology of Inflammation As Applied to the Gingiva

By HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

THE GINGIVA is composed of white fibrous connective tissue covered with stratified squamous epithelium. The epithelium of the oral mucosa thickens as it approaches the gingival margin, by great extension of the rete pegs inclosing finger-like papillae of connective tissue, each papilla containing a nutrient artery and vein with capillary arborization. The surface of the gingival epithelium has normally a cornified layer which extends over the margin and terminates at or near the margin of the sulcus. The lining epithelium of the sulcus is not so thick as the epithelium on the outer gingival surface, has little or no cornified layer and its rete pegs are much shallower. The connective tissue papillae may come very near the surface and in many specimens gaps, according to Box, can be found in which a papilla end comes to the surface and is uncovered by epithelial cells. Such breaks also occur frequently at the depth of the sulcus where the soft tissue is attached to the tooth surface. The epithelium normally extends beyond the depth of the sulcus to form a thin layer of cells in a band about the tooth, called the epithelial attachment. This band may be narrow and extend beyond the sulcus a short distance only or in cases of healed areas where periodontal pockets formerly existed, it may extend several millimeters along the tooth root. A thin layer of hyaline keratinous material is deposited by the epithelial attachment cells to form a cover. This adheres to the tooth structure when the epithelium is otherwise stripped away and remains as the dental cuticle. In areas not subject to abrasion this cuticle may remain for years. The connective tissue of the gingiva is coarse in the deeper layers consisting of heavy fibers for attaching the gingiva to the alveolar crest and to the cementum and attaching each tooth to its neighbors for support. The superficial connective tissue of the corium consists of a dense mass of fine fibers which give bulk and shape to the gingiva and serve the purpose of binding the gingiva tightly to the tooth surface. A few elastic fibers are present throughout the gingiva and periodontal membrane.

The blood supply to the gingiva is abundant consisting of anastamosing vessels from pericementum alveolar bone and oral mucous membrane. The abundant blood supply and the deep rete peg structure combine to make healing of the gingiva extremely rapid. A cut or abrasion usually heals completely in a few hours. Another peculiarity of the gingiva as contrasted to the skin is that the application of mild nervous stimuli such as mild heat or cold, pressure or friction does not cause a temporary hyperemia. The gingiva is nervously not a sensitive tissue. Unless hypersensitive due to inflammation, there is little pain when it is superficially cut, scraped, or compressed.

The gingival attachment gradually recedes during the lifetime. For some time after the tooth erupts, the base of the gingival sulcus is on the enamel. Later it may be at the amelo-cemental junction and still later some distance down on the cementum. In mouths which are kept healthy by tooth cleansing and gingival massage without abrasion of the gingival surface, the recession beyond the amelo-cemental junction is usually slight. Where recession exists, there is a cause for it. In the normal mouth kept healthy during the lifetime, there should be very few areas of cementum exposure indicating recession of the line of gingival attachment. Recession of this line means that the epithelium has proliferated toward the apex at the expense of periodontal connective tissue fibers. If the periodontal connective tissue is kept healthy by vigorous function and freedom from inflammation, it should be very resistant to encroachment by epithelium. Apparently it is, for many old persons who have had good mouth care show very little recession of the gingiva or abnormal depth of gingival sulci. Continual abrasion of the surface epithelium as in scrubbing the gingiva with a hard tooth brush causes gingival marginal recession. This is accompanied by a corresponding recession of the line of epithelial attachment at the expense of pericemental fibers. The

biologic mechanism of this recession does not appear to be as yet understood.

The normal gingival sulcus varies in depth on different portions of the tooth and also with age and between individuals. On the labial surface of an erupting tooth, the sulcus depth may be practically nil, the line of attachment almost coinciding with the margin. In most mature individuals it is approximately a millimeter deep on buccal, labial, and lingual and somewhat more in the interproximal areas. In cases which have had periodontal pockets that have been properly treated, the gingival sulci may be of much greater depth than normal (gingival pockets) and yet be in a condition of good health. An abnormally deep gingival sulcus (pocket), not due to gingival hypertrophy, means that destructive conditions have caused detachment of periodontal fibers. If the destructive condition is of an inflammatory nature then exudate and deposits on the roots will form and the gingiva will be loosened. When the inflammation is eliminated by sanitary measures and physiologic stimulation of the gingival tissue, the area will become healthy and no further destruction of periodontal tissue need take place. But physiologic reattachment of the epithelial lining of the periodontal pocket does not always take place even after excellent treatment, in which case a much deeper than normal sulcus (pocket) may be detected by probing. Deepened sulci (pockets) even when they involve bifurcation areas, if they can be kept healthy by cleanliness and gingival stimulation do not necessarily indicate an unfavorable prognosis.

The gingival sulcus around the tooth offers an unfavorable anatomical condition under modern conditions of living. Under conditions of wild life in which food is eaten raw, in a coarse state, and in limited amounts, the teeth are kept clean and the gingival and periodontal tissues are kept healthy and vigorous by the massage of the coarse food against the gingiva and by the vigorous function of the teeth themselves. Periodontal disease, according to Colyer is limited in wild animals to the effects of foreign bodies which in one way or another become lodged under the gingiva and lead to infections. Domesticated animals fed on an excess of food which requires little function of the teeth and jaws may develop disease of the gingiva due to accumulations of debris which adhere to the teeth against the gingival margin. Inflammation of the gingiva in turn may lead to periodontoclasia and gingival recession in dogs, cats, and other animals in captivity in the same way as it does in modern human beings.

The debris which forms on the teeth is partly from the saliva, partly from exfoliated gingival epithelial cells, and may be partly from food remnants. The material is organic in nature and becomes a culture medium for the active growth of bacteria and fungi of the mouth. The mass is highly irritating to living tissue in contact with it. The marginal gingiva may be irritated and inflamed by this debris in which case it will be loosened to increase the sulcus space. Also the gingival lining of the sulcus will give off exudate to add to the intensity of bacterial growth and formation of locally poisonous products.

The organic accumulations on the tooth surfaces against the gingival margin may or may not easily cause inflammation of the marginal gingiva depending on factors many of which are not yet known. Of the known causes for great resistance undoubtedly active function is the most important. Waugh has shown that primitive Eskimos have very dirty teeth with heavy accumulations of putrefying soft deposits and salivary calculus, yet practically never have periodontal pockets. The gingivae may recede due to the accumulations but the effect is rather atrophic than inflammatory. He attributes this phenomenon to the very great function to which the teeth are subjected.

Some of the endocrine hormones also modify resistance by their effects on the epithelium of mucous membranes. Ziskin has shown that estrogens and testosterone have the effect of stimulating an epithelium with a well developed cornified layer which is very resistant to infection or the penetration of irritating chemical substances. On the other hand, an anterior pituitary-like hormone believed to be derived in most part from chorionic cells of the placenta, causes denudation of the cornified layer of the gingival epithelium leading to softened and reddened marginal gingivae. Such gingivae are easily penetrated by bacteria and toxins to cause inflammation. This results in the so-called pregnancy gingivitis.

There is evidence that some of the vitamins may play a part in the resistance of the

marginal gingiva to the effects of irritating deposits on the teeth. Nutritional studies indicate that gingivitis may be associated with certain nutritional deficiency states and may be corrected by fortifying the diet. Hanke has produced evidence which suggests that abundant vitamin C is beneficial as a preventive and curative agent for gingivitis in children and that subclinical C avitaminosis is associated with gingivitis. King has evidence to suggest that niacin deficiency is associated with susceptibility to the Vincent's type of gingivitis and that niacin may be used as a curative agent. While there is much enthusiasm among dentists regarding nutritional deficiencies as predisposing causes of gingivitis, Vincent's infection, and periodontoclasia, and wide spread prescribing of vitamins for these conditions, the evidence on which this idea is based is as yet pretty meager and uncertain.

The clue to the destructive nature of gingivitis lies in the physiology of inflammation. According to Menkin:

> Inflammation may be broadly defined as the complex vascular, lymphatic, and local tissue reaction elicited in the higher organisms by the presence of microörganisms or non-viable irritants. It represents a basic or elementary reaction to injury whereby the deleterious agent tends to be localized and ultimately destroyed.

By such localization the organism is protected at the expense of destruction in the local tissue.

As soon as tissue cells are injured, they liberate a stimulating substance which Menkin calls "leukotaxine." This chemical messenger affects the capillaries in the immediate region to cause them to be permeable to proteins. It also is positively chemotactic to P.M.N. leukocytes and causes them to collect in the neighboring blood vessels and pass through into the tissue spaces. Fibrinogen escapes into the tissue spaces along with other protein molecules and forms a fibrin network that tends to confine bacteria or other particles from extending into new tissue. The P.M.N. leukocytes secrete proteolytic enzymes which in time dissolve tissue elements, bacteria, and the leukocytes themselves into liquid pus. When sufficient liquefaction has occurred to open to a surface the pus is evacuated thereby eliminating the foreign invading body, providing it can be dissolved by the leukocytes. The process is one in which the organism as a whole is saved from overwhelming invasion at the expense of the local tissue.

The peculiar conditon in gingivitis lies in the fact that an irritant is present against or under the gingival margin which causes inflammation of the gingival tissues, but the irritant remains outside the body integument where it is inaccessible to the fighting forces of the body. The destructive effects of inflammation destroy the gingival and periodontal tissues without in any way eliminating the cause of irritation. The inflammation, by loosening the gingiva and by exuding protein containing fluid into the sulcus, rather adds to the cause of trouble by furnishing space and food for increased growth of microörganisms.

The first effect of tissue injury is the reaction of the capillaries which become permeable so that blood proteins pass out into the tissue spaces. The physical chemical mechanism for interchange of fluid between the capillaries and the tissue cells is a nice one. The capillary is a small tube composed of a single layer of endothelial cells. Outside the endothelial cells, the cells of Rouget are placed at intervals with retractile tendrils which wrap around the capillary. The cells of Rouget are supplied by sympathetic nerves which upon stimulation cause the cells to contract. When these cells contract, the capillary closes as a tube and blood ceases to flow through it. The stimulus to relaxation and opening of the capillary seems to be from a chemical substance liberated in the tissue either by the cells themselves when underoxidized or injured, or by action of autonomic nerves which come to the tissue but do not directly reach the cells of Rouget.

The presence of protein in the fluid within the capillary which cannot escape through the capillary wall while water and small molecule salts and amino acids diffuse freely, creates osmotic force which tends to draw fluid from the tissue spaces into the capillary. This force is offset by the capillary blood pressure which is greater at the arterial end than the osmotic force but diminishes along the length of the capillary until it is less. Thus fluid is forced out of the capillary on its arterial end and forced in on the venous end. The two actions are balanced to create optimum food and drainage conditions in the tissue.

This beautiful balance of forces is destroy-

ed when inflammation causes an increase in capillary permeability. The proteins then escape from the capillary into the tissue spaces and the osmotic force which should maintain the fluid content of the capillary is lost. The capillary is left plugged with red blood cells and no longer functions as a carrier of blood for tissue fluid interchange. Fluids escape into the tissue spaces and cannot get back. The tissue becomes waterlogged or edematous.

The plugging of the capillary circulation which occurs with inflammation reduces the oxygen carbon dioxide interchange. The tissue becomes underoxidized and gains a purple hue. Insufficient oxygen and accumulated CO_2 increase the hydrogen ion content of the tissue for it permits the accumulations of acid metabolites. Inflamed tissue may become acid in reaction whereas normal tissue is always slightly alkaline with pH 7.1 to 7.4. The effect of acid on collagen of which white fibrous connective tissue is composed, is swelling, softening, and final solution.

P.M.N. leukocytes are attracted by the leukotaxine liberated by injured tissue cells. Leukotaxine according to Menkin has been crystallized and purified but has not yet been completely identified. By chemical tests it is a polypeptide and not histamine or a histamine-like substance. When leukotaxine is liberated in the tissue, the capillary walls of the region will be lined within fifteen to twenty minutes with P.M.N. leukocytes. Within thirty to forty minutes these leukocytes will have worked their way through the capillary walls and be found in the tissue spaces.

P.M.N. leukocytes are killed or repelled when the pH gets below seven, which is neutral. As the tissue becomes underoxidized and progressively more acid the P.M.N. leukocytes disappear. This disappearance is probably due to the accumulation of lactic acid in the tissue which is negatively chemotactic to P.M.N. leukocytes; that is, repels them. It is a stronger repellent than leukotaxine is an attractor.

Monocytic leukocytes are not killed until a pH of 6.7 or lower is reached. When the tissue is acid, lymphocytes and plasma cells predominate. Inflammation is not necessarily conducive to a state of local acidosis. If not, the P.M.N. leukocytes will continue to predominate. Menkin says pus forms when acidity becomes sufficient to kill all leukocytes.

It is not acid alone that dissolves the collagen of connective tissue. The P.M.N. leukocytes secrete a powerful protease which acts to dissolve the tissue elements. Collagen solution will therefore take place in the presence of large accumulations of P.M.N. leukocytes while the reaction is still alkaline. Autolytic enzymes are liberated when the tissue reaction becomes acid and aid in the solution of collagen and tissue cells.

Applying these facts to the gingiva inflamed by the presence of a mass of irritating material against or under the margin, the clinical aspects of gingivitis and periodontoclasia become understandable. The first sign is edema due to capillary permeability. This shows itself as a shiny slightly rounded margin. The shininess is due to the fact that the surface epithelium is stretched until it has lost the uneven corrugations which give it a stippled appearance. The second sign is a change in color to a deeper red or a purple hue. This color change indicates capillary stasis with accumulating CO_2 and deficiency of oxygen.

Histologic examination shows accumulating lymphocytes in the gingival tissue of most individuals which indicates an acid reaction of the tissue with repellence of P.M.N. leukocytes. In individuals with high alkaline reserves, the tissue may not become acid in which case P.M.N. leukocytes will be attracted and will enter into the gingival sulcus to form white pus. White pus indicates an alkaline tissue reaction to an irritant. Its absence does not necessarily indicate a condition of health.

Inflammation will not exist long in the marginal gingiva without loosening of this tissue. The gingiva no longer forms a tight cuff surrounding the tooth but becomes flabby so as to widen the gingival sulcus. This sign means that the white fibrous connective tissue of the gingiva is swollen, softened and disintegrating. When this disintegration has gone to the extent of involving the pericemental fibers just beyond the epithelial attachment then a proliferation of epithelial attachment occurs along the cementum to take the place of the disintegrated fibers. This explanation is somewhat at variance with that of Gottlieb who attributes the advance of epithelium along the cementum to a death of cementum or rather

a failure to form new cementum layers continuously. Since the formation of new cementum layers is a function of the pericementum, the fault in either case seems to be a failure in the pericementum to maintain its function and structure, due to changes in the chemistry of the tissue which in turn are the result of capillary stasis.

The gradual destruction of the pericemental tissue with encroachment of epithelium along the cementum apically is undoubtedly the way most periodontal pockets progress. A flabby tissue attached to the tooth surface by a deep layer of epithelium is of course easily torn from the root surface by all sorts of mechanical injuries. The epithelium lining a sulcus which is filled with irritating material is also changed and may be easily split or chemically destroyed to deepen the sulcus into a pocket.

This epithelial change is graphically portrayed by Box in 1942 in an article on the "Formation of Subgingival Calculus". In the pictures which are shown, the epithelium at the base of the gingival sulcus which was most intimately in contact with the irritating deposit had completely lost its staining properties. The basement membrane cannot be distinguished and the nuclei and cytoplasm of the cells are so degenerated that it is impossible to make out where living tissue ends and dead deposit begins. A deep layer of necrotic epithelium certainly would not long remain attached to the root surface.

Box has shown in this and other articles that the epithelial lining changes due to deposits in the gingival sulcus consist of ulcerations which extend through the epithelium into the underlying connective tissue, of gaps in which connective tissue papillae reach the surface and of areas of necrotic epithelium. Even when the epithelium is thick and vital, its cells are separated and loosened by inflammation and edema.

The result in any case is the escape of tissue fluid into the gingival sulcus. When capillary permeability is established and protein escapes into the tissue spaces then the exudate will contain protein. The protein containing exudate now becomes the chief source of culture pabulum for the bacteria and fungi that have established themselves in the widened gingival sulcus.

Box in the 1942 article shows that calculus appears to form about the bodies of actinomyces-like masses of fungi. This suggests that these microörganisms change the environment about themselves in such a way as to bring down calcium and phosphate ions as the apatite salt. This may come about by the secretion of an enzyme like phosphatase which brings phosphate out of organic into ionic form. Or it may form alkaline products as a result of its action on the protein material and thus create an alkaline reaction about itself which brings the calcium and phosphate ions out of solution.

The source of the ions may be from the saliva if the gingival sulcus is shallow and easily accessible to the saliva. In the gingival pockets the exudate is undoubtedly a much more prolific source. Not only does the serum have a fairly high concentration of calcium and phosphate in inorganic ion form but also it contains calcium combined with protein, and phosphate combined in various organic forms, all of which escape into the tissue fluid and thence into the exudate when the capillaries are permeable. When the bone margin becomes involved in the area of hyperemia then the bone is demineralized; that is, its salts are removed from the organic bone matrix and are added to the tissue fluid and exudate. The gingival exudate is thus a rich source of calcium and phosphate ions. Many deep pockets with narrow orifices have heavy calculus masses at their depths which are difficult to explain on the basis that the ions came from the saliva.

One of the interesting experiments of Menkin related to the collection of dye which had been injected intravenously into an animal, in areas in which capillary permeability had been produced by one means or another. The edematous spots on the skin were brightly colored by the dye which was otherwise not visible. The explanation is that the large dye molecules are able to escape from the capillaries which have become permeable so that the dye which was injected tends to collect in the affected area whereas it does not ecsape into the tissue spaces in normal body tissues.

This experiment gives a clue as to why mercury, bismuth and lead salts, dilantin, and some other chemicals tend to collect in edematous gingivae until they have concentrated in poisonous quantity. If the gingivae are kept free of inflammation and edema, it has been demonstrated by periodontists on many occasions that these substances have no special predilection for the gingivae. It has been shown furthermore that by reducing the gingival edema with proper treat-

ment, the local tissue will rid itself of its load of foreign material and become healthy during the time that the drug is being continued in systemic administration.

The role of bacteria which invade the gingival tissue in gingivitis and periodontoclasia is not clear. The microörganism found by study of sections and by culture deeply penetrating into the tissue is the *Streptococcus viridans*. Other microörganisms are not found to penetrate or invade appreciably. The *Streptococcus viridans* is unusually well adapted to live in the tissues of the human host but under normal conditions it does not do much damage to tissue or cause much reaction in the tissue.

In cases of marked gingivitis or periodontoclasia or chronic periapical granuloma these bacteria will so infiltrate the tissues, even working their way into the capillaries, that any manipulation of the tissue will cause a transient bacteremia that can be detected by blood cultures. In the course of a few minutes the blood again becomes bacteria free, indicating that the bacteria in it have been screened out probably by the spleen and by the endothelial cells lining the blood vessels. It is only in cases of a previously damaged or anatomically defective heart, that the lining epithelium of the heart becomes infected by these organisms, presumably via a bacteremia from some locally infected tissue causing subacute bacterial endocarditis.

The *Streptococcus viridans* that has invaded the chronically inflamed, edematous, clogged gingiva is evidently merely taking advantage of the damaged state of the tissue to invade it. It does not appear to be responsible primarily for the inflammation nor greatly to hinder its recovery once the irritating deposit on the tooth surface is removed. With this situation in mind, it is probably accurate to say that infection is secondary rather than primary in gingivitis and periodontoclasia. These diseases are due primarily to deposits lying against the gingival tissues which are irritating because of the microbic life within the deposits. The bacteria which render the deposits so irritating are probably entirely saprophytic and non-invasive.

Tenenbaum and Karshan have recently demonstrated that many cases with considerable periodontoclasia exhibit a high sedimentation rate. They have been experimenting on a large number of clinical pathological tests in these cases in the effort to find some systemic chemical irregularity to account for supposed susceptibility to periodontoclasia. So far no test has given consistent results except high sedimentation rate which is used as an indication of inflammation somewhere in the body. It appears from their work that gingivitis and periodontoclasia, if considerable in amount, do exert a deleterious influence on the body that may be tested chemically. It is the common experience that persons with severe periodontoclasia are not in good health in some respect. When the local inflammation has been subdued and the oral tissues brought into health, the patients generally experience an improvement in general health. Whether this benefit is related to freedom from toxic products of inflamed tissue or to freedom from bacteremia is not known at present.

Treatment of gingivitis and periodontoclasia consists in removing the irritating deposits on the tooth surfaces with steel instruments and building up the health of the gingival and periodontal tissues by massage. Accessory irritants like traumatic occlusion and food impaction are of course discovered and eliminated by appropriate clinical measures.

The restoring to health of the gingival and periodontal tissues by massage is as interesting a physiologic process as the steps by which the tissue broke down. We start with a tissue hanging loosely around the tooth, the lining epithelium of the pocket degenerated, off in patches and exuding. The underlying connective tissue fibers are swollen and softened, the tissue is edematous with fibrin, protein, salts, and water in the tissue spaces, the bone demineralized at the alveolar margin and in advanced cases considerably destroyed, the capillaries permeable and plugged with red blood cells, and *Streptococci viridans* scattered throughout the tissue.

Gingival massage is a series of compressions and releases of the gingival tissue. When the tissue is compressed, it blanches which means that blood is forced out of it. The capillaries filled with packed red blood cells are emptied into the veins. When the pressure is released the blanched tissue turns pink or red which means that the emptied blood vessels are again filled with blood which is fresh from the arteries. When the tissue cells are no longer irritated by the root surface deposit, they no longer give off

leukotaxine and the capillaries resume their normal tone and become impermeable to proteins. The fibrin and proteins in the tissue spaces become digested by ferments from the cells and disappear. The epithelium being no longer poisoned on one side and starved on the other resumes its normal covering function so that exudate stops flowing into the gingival pocket. The connective tissue again assumes its function and tightens so that the gingiva is bound tightly to the tooth surface. The epithelium lining of the pocket may or may not attach itself to the root surface. If so, the pocket is eliminated to the extent of the attachment. If not, the gingival tissue hugs the tooth so tightly that there is practically no space for the further collection of deposit. The bacteria gradually disappear from the tissue.

The bone builds in so far as matrix remains for it to build into. This means clinically that bone margins which have faded out and disappeared in the radiograms now show a dense cortical plate passing over the margin from one lamina dura to the next. Bone should not be expected to grow back where once a pocket existed. The epithelial lining of the pocket does not disappear and become replaced with new periodontal membrane and cementum under the conditions resulting from periodontal treatment as usually practiced. Whether such a result can be often attained by changes in techic aimed at eliminating lining epithelium in the pocket and stimulating rapid bone and cementum formation in the area is still a matter for the future. In most instances it is sufficient to place the remaining tissues in health. If half the tooth root is attached and can be maintained in health, it is enough to permit normal functioning of the tooth with few exceptions.

To accomplish these purposes, it is necessary that the pressure of the massage reach the deepest portions of the tissue affected by the capillary stasis. It may require some ingenuity to devise means for compressing tissue in narrow spaces between roots or in inaccessible bifurcation areas or spots where cheek, ramus, or tongue prevents access with the tooth brush.

Massage is a mechanical pumping of the blood into the tissue. Its effect is not to be obtained by irritant drugs applied to the gingiva or by stroking the gingiva with the tooth brush. What is desired is compression applied in the apical direction to the gingival margins. In most instances this may be obtained by the proper use of a hard bristle brush, the bristle ends being applied to the tooth surfaces and the sides of the bristles brought with pressure against the gingival margins. Any scrubbing of the gingiva with bristle ends is to be avoided. Where deep involvements have existed especially on the buccal or lingual sides of the tooth roots, finger massage may be added as a means of compression. If involved tissue cannot be reached by the pressure of massage, it should not be expected to heal. Once the gingival tissue is seriously affected, it is not enough to remove the deposits. Without the artificial respiration of massage the healing process is so slow that before the gingival tissues have tightened a new aggregation of deposits will have formed. If an area is inaccessible to massage then surgical resection of the congested tissue would appear to be a last resort if the affected tooth is to be saved in health. An understanding of the physiology of the affected tissue and of the value of massage in restoring health should make a resort to surgery seldom necessary.

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BOOK REVIEWS

Oral Histology and Embryology. Edited by Balint Orban, M.D., D.D.S. (St. Louis, The C. V. Mosby Co., 1944). 342 pp. 262 illustrations.

Orban's *Oral Histology and Embryology*, published this year, is an admirable work of collaboration by nineteen specialists. The plan of collaboration employed represents a new idea in coöperative literary endeavor. A single contributor was responsible for the original draft of each chapter or section, but this version served only as a basis for criticism and revision by all the other contributors. In this way it was planned to harmonize variant opinions; and in general, as a result, a unity was attained seldom achieved by a collaborating group. It is noteworthy, however, that those parts of the text which deal with the traditionally studied tissues—such as the section on enamel and the chapter on dentin—are less positively presented than are the parts concerned with structures which have not been so thoroughly examined.

The scope of the book distinguishes it from works previously published to which it might properly be compared. The whole mouth, rather than just dental structures, is presented; and emphasis is laid on the physiology and development, rather than on structure, although the latter is adequately presented. From the time of John Tomes' *Dental Physiology and Surgery*, in 1848, there has been a trend away from odontology toward stomatology, as may be seen in the revision of Noyes' *Dental Histology and Embryology*, in 1938. The earlier books discussed only the hard tissues of the teeth, while the later ones included supporting and neighboring structures. Orban's new book goes further in this direction. The broader approach of the new work is indicative of the increased demand on modern dentistry to know the mouth as a whole organ and not merely the structure of its component parts.

Every chapter includes a section on clinical applications of the knowledge presented. These passages are in sharp contrast to the scientific attitude and almost academic tone of the rest of the text. Such attempts at correlating "theory" and "practice" are justified in a work of this kind, be it used by undergraduates or by post-graduates and graduates as a reference work.

The illustrations are uniformly good with the exception of those purporting to show enamel rod direction in deciduous and permanent teeth as it relates to Black's principles of cavity preparation. Lettering illustrations would effectively eliminate difficulties arising from uncertain terminal points of lines in the illustrations. The long debated question involving the possible vitality of the cementum and the repair capacities of the periodontal membrane, with its contained circulation, has not been conclusively settled. There is also no conclusive proof that teeth drift mesio-occlusally; some do and some do not. They may drift in many directions, depending upon the stresses and strains produced by the functioning of the mouth.

—G. B. Denton and G. R. Lundquist.

The Dental Treatment of Maxillo-Facial Injuries with Supplementary Material on Cases and Techniques. By W. Kelsey Fry, Consulting Dental Surgeon to the Royal Air Force, Consulting Dental Surgeon to the Ministry of Health, Dental Surgeon to Guy's Hospital, London, England. P. Rae Shepherd, Dental Surgeon, East Grinstead Maxillo-Facial Unit. Alan C. McLeod, Dental Surgeon, East Grinstead Maxillo-Facial Unit. Gilbert J. Parfitt, Dental Surgeon, East Grinstead Maxillo-Facial Unit.

With foreword by F. R. Frazer, Director General, Emergency Medical Service; and a section on Fractures of the Middle Third of the Face by A. H. Mc Indoe, Consulting Plastic Surgeon to the Royal Air Force, Surgeon in Charge, East Grinstead Maxillo-Facial Unit.

Cloth 428 pages, 843 illustrations inclusive of drawings and plates. Index, table of content with cross references supplied in the text as well as at the top of pages.

American edition by J. B. Lippincott, Philadelphia and Montreal.

This volume contains all the material as covered in the review for the Supplement to the July 1943 issue of this Journal together with the Supplemental volume subsequently published by the authors. All of which makes for a more complete presentation of this most important subject.

The Foreword to the American Edition was written by one who evidently saw the group at work. A man whose training and position well qualifies him to speak with authority. This foreword is herewith quoted:

This straightforward presentation of the authors' work has appeared at an opportune time. With the advantage of central direction, concentration of patients, open discussion and evaluation of results, they have put their conclusions down clearly.

The plan of coöperation of the dental surgeon and the maxillo-facial or plastic surgeon will produce the best results, and the care and consideration of the patient is one of the main essentials of the book. Although it is modestly stated that the book is 'primarily intended for those dental surgeons who have had little or no experience in the treatment of these injuries and who may be called upon to treat them at any moment,' it is so well written and contains such sound knowledge of fundamentals that it can be very valuable to all who are interested in this work.

The senior author started in the last world war, where he came honestly by first-hand information, having been injured twice in forward work. He has carefully evaluated the situation, has worked closely with his associates, and with open minds they have settled on this final procedure. The spirit of coöperation in the workers has been a pleasure to see and it extends right into the book—so that the book is readable, clear, interesting, and the very lack of expansive writing naturally puts the reader on the side of the authors.

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Army of the United States:
Senior Consultant in Plastic and Maxillo-Facial Injuries and Burns, E.T.O., U.S.A.

No further remarks seem necessary to emphasize the importance of this volume.

—*Walter M. Morgan*

Oral Pathology: A Histological, Roentgenological, and Clinical Study of the Diseases of the Teeth, Jaws and Mouth. By Kurt H. Thoma, Second edition: pp. 1328, 1388 illustrations. [Review reprinted from] *J.A.M.A.* 125 (6): 458. June 10, 1944.

This edition, issued shortly after the first, represents an increase in text to include a few of the rare diseases with the addition of new illustrations and the replacement of others. By stressing pathologic processes the author tries to create a visual picture of microscopic changes in diseases and correlate them with the clinical symptoms and the roentgenologic findings. This type of descriptive approach of clinical entities, often neglected in the average textbook, is emphasized throughout and, with the profuse illustrations, furnishes an excellent reference book for the diagnostic study of the patient by the clinician, for the interpretation of the X-ray illustrations about the head by the roentgenologist, and of biopsies about the mouth by the histopathologist.

The numerous illustrations of patients, X-ray appearance of pathologic processes are instructive and well arranged. Those in color represent color photographs and photomicrographs. In a few instances, the detailed histopathologic changes are insufficiently reproduced to correspond to the descriptive text.

The first portion is concerned with hereditary, endocrine, vitamin and other nutritive influences on the development of the teeth and jaws. Systemic and local factors are discussed, with their effect on growth and structure. Functional and morbid changes under various physiologic and environmental conditions are discussed. Local experimental factors on teeth and gums is an interesting chapter. Dental caries particularly from the investigative point of view, is well described; however, focal infection in odontogenic infection is disposed of briefly. The relationship of blood dyscrasias, deficiency diseases, metabolic disturbances, allergy, and virus infection to lesions of the mouth are worthy of special emphasis.

The excellent bibliography appended at the end of each chapter includes medical as well as dental literature, together with important foreign contributions. While the book is primarily written for the dental profession, the medical practitioner will appreciate the large volume of general information that emphasizes the recent work in the investigative and pathologic phases of diseases of teeth and jaws as well as the illustrative chapters of lesions of the oral mucosa, lips and tongue. This volume will fill an important place in a medical laboratory.

Fundamentals of Dental Histology and Embryology Including Clinical Applications. By Charles F. Bodecker, D.D.S., F.A.C.D., Professor of Oral Histology and Embryology, School of Dental and Oral Surgery, Columbia University, New York.

87 illustrations. Published by Columbia University Press, New York.

In recent years, ever increasing emphasis is being placed upon the biological aspect of dental practice. This book is written with the object of giving such knowledge that the dentist may be guided by biological principles. Information is conveyed chiefly by means of diagrams rather than by extensive text. The author believes that a painstaking sketch will leave a more lasting impression than an endless repetition of a worded description. Besides a discussion of the minute structure of the oral tissues, some references are given concerning the practical application of histology to the clinical practice of dentistry.

This fourth edition includes many new illustrations, including a few three dimensional drawings in order to give this concept of the dental tissues. This enables one to visualize the relationship of the instruments used in different dental operations to the microscopic anatomy of the teeth and surrounding tissues.

Teeth are a part of a vital organism and so histology is of real practical value since without a knowledge of this subject, one can never understand pathology. It is of great importance to know the minute structure of the tissues we operate upon if we are to recognize cause and effect of the various conditions one observes in general practice.

This book is to be highly commended to all those who wish to practice dentistry upon the highest possible plane.

—*M. H. Garvin*

[*Reprinted from Jour. C.D.A.* 10(8):363. Aug., 1944]

Hackh's Chemical Dictionary: (American and British Usage). Containing the words generally used in Chemistry, and many of the terms used in the related sciences of Physics, Astrophysics, Mineralogy, Pharmacy, Agriculture, Biology, Medicine, Engineering, etc. Based on recent chemical literature with numerous tables, diagrams, portraits and other illustrations. Third Edition. Completely revised and edited by Julius Grant, M.Sc., Ph.D., F.R.I.C. . . . . Pp. XII+925. [Philadelphia: The Blakiston Company, 1944]

Hackh's Chemical Dictionary is an authoritative publication which has met with world wide acceptance. The present edition, the third, has been revised by Doctor Grant alone, Ingo Hackh having passed away in 1938. Grant, who collaborated with Hackh in the earlier editions has continued along the objectives and scope of the dictionary as set down in the earlier editions with the exception of the omission of pronunciations. In revising the dictionary attention has been given to balancing the treatment from the American and British points of view.

The dentist who is interested in the metallurgy and physical properties of the material he uses and the research worker in this field will find here a valuable dictionary since products are not only defined but their physical and chemical properties and symbols have been included. Thus, the acrylic acid compounds, penicillin, the sulfa drugs and other new chemicals, pharmaceuticals, metals and compounds are mentioned.

[Reprinted from N. Y. J. of Dent. 14(12):381. 1944]

Parodontal Disease. By E. Wilfred Fish, M.D., Ch.B., L.D.S., D.D.Sc. (Melb.) will be reviewed in the April issue of this Journal. [Ed.]

The Canadian Research Foundation announced early in 1944 another Bulletin by Harold K. Box, Number 25.

Necrotic Gingivitis; Its Histopathology and Treatment with an Adherent Dressing. To be reviewed later.

Time to Revise Fees

[*Reprinted by Permission of the Editor of The Fortnightly Review 8(4):7. Aug. 15, 1944*]

THE HIGHEST fees which the average dentist charges are for prosthetic appliances, the construction of which requires a minimum of his time if he uses the laboratory. The treatments which require most of his operating time are too often those for which his fees are lowest. In this respect dentists have followed the path of least resistance; it is easier to charge for tangible objects such as material and appliances which patients can see and feel than it is to charge for intangible services which may save teeth and preserve health but which are not readily apparent to patients. Fee schedules become established in a community and dentists tend to follow them indefinitely; they hesitate to deviate far from the standard.

The public judges value by the price it pays, the more the cost the greater the appreciation. The high prices for the replacement of natural teeth has tended to minimize the importance of other services which save teeth. The result has been undue emphasis on replacement service; in fact repairable natural teeth are sacrificed so that good looking replacements can be made. We do not mean to minimize the health value of prosthetic restorations and the importance of aesthetic appearance, but the fees for other dental services do not reflect in proportion their importance.

This disproportionate fee schedule has magnified the mechanical side of dentistry and minimized the biological; many regard dentistry as a mechanical trade and not a profession founded on an understanding of biology. Actually the dental student spends a large portion of his college hours acquiring an understanding of the fundamental sciences and their relation to the oral cavity. The dentist spends most of his time performing operations based on that knowledge and directed toward retaining natural teeth. Everyone will agree that natural teeth properly preserved are better than the best artificial substitutes.

Diagnosis and treatment planning, cavity preparation and filling, prophylactic and preventive measures, treatment of soft tissues and patient education are time consuming procedures. How often are root canals treated for little or no fee so that expensive restorations can be placed. Yet root canal therapy properly done requires time and represents the foundation for the restoration. The payment for the less tangible services usually are hidden in the fee for the prosthetic restoration. This procedure may have been expedient but it misleads the public and especially those who may be seeking to change dental practice.

It is time for dentists to get better fees for the services which they have

spent years in mastering, which only they can perform by right of their training, and which preserve the natural teeth for years of safe, comfortable function. Official bodies in organized dentistry should take cognizance of this problem and analyze methods for its correction in the post war period immediately ahead. *Editorial—Robert G. Kesel.*

The next "In Our Opinion" Discussion will be published in the April, 1945 issue. Subject: "The Use of Drugs in Periodontia Practice."